CLINICAL PROCEEDINGS

of the

CHILDREN'S HOSPITAL

WASHINGTON, D. C.

December 1951

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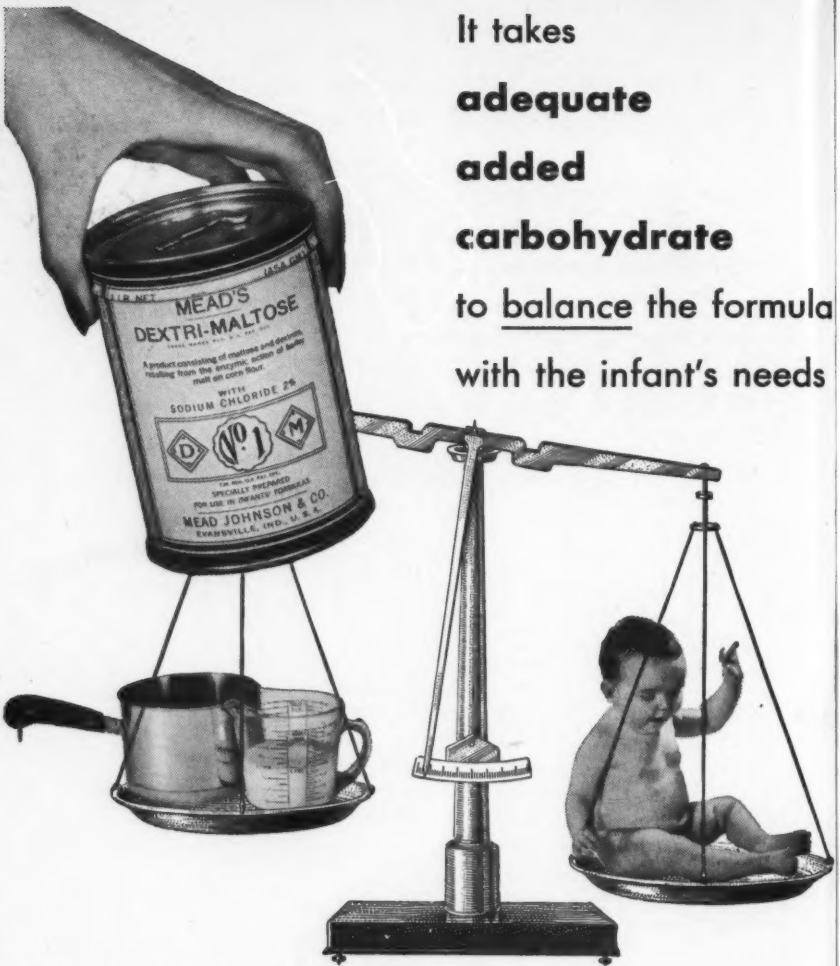

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CLINICAL PROCEEDINGS

OF THE CHILDRENS HOSPITAL

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CONTENTS

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DEDICATION

Joseph Stiles Wall, M.D.

Edgar P. Copeland, M.D.

On September 10, 1951, Dr. Joseph Stiles Wall resigned from his position, Chairman of the Medical Staff of Children's Hospital, Washington, D. C. At Dr. Wall's request the following letter was presented to the staff:

Dear Dr. Copeland:

Would you be good enough to present to the Medical Staff at its meeting on September 10 this letter of resignation as Chairman of the Staff to take effect October 1, 1951?

This date marked the anniversary of my beginning association with the Children's Hospital as Resident Student fifty-five years ago on October 1, 1896.

After this long service I feel that I might be entitled to be relieved of my active duties with the hospital.

I would be remiss if I did not express my great reluctance to terminate active service with the hospital which for over half a century has given me such pleasure and gratification and I would take this occasion to indicate my deep appreciation of the cooperation, loyalty, and affection the members of the Staff have constantly afforded me throughout the past years.

Sincerely,
Joseph Stiles Wall, M.D.

To record the resignation of Dr. Wall as Chairman of the Staff of Children's Hospital brings sadness to us all. For fifty-five years he has served the Hospital with a devotion beyond compare. As Chairman of the Staff since 1938, he brought to the position administrative ability and a dignity that won the loyalty and admiration of all with whom he came in contact. His leadership was an inspiration that welded the members of the Staff into an harmonious group, zealous in accomplishing the ideals for which he stood. During his tenure of office, significant and constructive changes have been made in the Hospital's organization that have greatly enhanced its reputation. New departments have been created that have expanded the Hospital's activities and increased its usefulness in the field of welfare.

Dr. Wall may well reflect upon a distinguished career, consecrated, as it has been, to the welfare of children and the public in general. His accomplishments have won him national and international recognition and the esteem and devotion of people in the community in which he has lived. It is no wonder that he wearies after his labors, but the Creator must have endowed him with special courage and endurance for his tasks.

The vacuum created by Dr. Wall's resignation will be keenly felt for a long period, but we are comforted by the thought that, as a member of

the Consulting Staff, his counsel will always be available when storm clouds threaten. With a full realization that he has well earned relief from his labors, we wish him health, happiness and long life in retirement from the arduous duties that he has relinquished.

For all that Dr. Wall has given us we, who carry on, are deeply grateful. At the direction of the Staff, the following letter was sent to Dr. Wall:

Dear Dr. Wall:

Sadness and surprise pervaded the atmosphere of the Staff Meeting yesterday when Dr. Burdick, the Secretary, read your letter of resignation as Chairman of the Staff.

It was accepted with a realization and a full appreciation of the condition that in your judgement made it necessary.

You have served us long, faithfully, and well and I am sure that you are conscious of our devotion and the esteem in which you are held. Your ability and your fairness as our presiding officer has inspired us at all times—and, while we are to be deprived of your presence, we shall always feel free to counsel with you as a member of our Consulting Staff.

The memory of our happy association will endure always and, as a token thereof, the Staff has unanimously nominated you to be permanent honorary chairman of the Medical Staff of Children's Hospital.

With our very best wishes for a speedy return to good health, we beg to remain,

Devotedly yours,
The Medical Staff
by Edgar P. Copeland, M.D.

ON THE EPIDEMIOLOGY OF ACCIDENTS IN CHILDHOOD

EDITORIAL

John P. McGovern, M.D.

The prevalence of accidents especially among children has been brought into focus recently by the reduction in the number of cases and the increased control of many infectious diseases. With the conquest of communicable diseases of children, accidents now represent the *leading cause of death*——actually one-third as many deaths as all diseases combined, in children from ages 1 to 14. In one year, over 12,500 children were killed by accidents. The actual number of children permanently crippled is completely unknown, but on the basis of a study by Roberts and Gordon, in which they found that for every person killed 150 were injured and 4 received permanent disability, one might infer that approximately 50,000 children a year are permanently injured in the United States. In an average year, accidents alone result in the death of more than 5,000 preschool children between the ages of 1 and 4 years.

Although one might surmise a higher figure, approximately one-fourth of these accidents are due to motor vehicles. Most of the other accidents occur in and around the home.

From the foregoing facts, the conclusion is inescapable that the greatest problem in preservation of lives of children is accident prevention, especially in the home.

It has become generally recognized that accidents seldom "just happen." They are nearly always caused by some definite factors. It has been said that 90 per cent of all accidents are preventable. One competent observer has expressed the opinion that four-fifths of all accidents to children under five years of age are due to errors of omission or commission by adults.

In the past, accidental deaths and injuries have been accepted as more or less inevitable and their prevention was considered to be outside the realm of science. A selected review of the literature (1940–1950) reveals a recent gradual change in this concept that has been marked by some little research, study, and action (chiefly in the areas of industrial and traffic safety). The success of some traffic safety programs in reducing child traffic fatalities typifies this mode of effort.

It is the opinion of this observer, however, that before any real progress can be made in resolving the home-accident problem, we must have more precise knowledge of the natural history of accidents and that this knowledge can best be gained through the epidemiological approach. Someone might insist that there is no epidemic of accidents at present. That is a debatable stand, for certainly we may well be in the midst of an epidemic of accidents at the present time as related to one hundred or 1,000 years ago. As our vital statistics for accidents do not go back for more than 50 years, one cannot conclusively prove this. It seems obvious, however, that as we harvest the forces of nature and convert them into technical advances, we are introducing more and more accident possibilities. For example, in the first 40 years of this century, deaths due to automobile accidents alone rose from 0 to 26.2 per 100,000.

In other words, it is quite probable that in the natural history of accidents, the current prevalence does represent an epidemic. Just what constitutes an epidemic? Webster says that an epidemic is "a rapidly spreading or widely prevalent attack of disease." Accidents are certainly widely prevalent. Exactly what is a disease? Is it only malfunction due to bacterial, viral, metabolic or metaplastic impairment? Again from Webster, a disease is "a condition in which bodily health is seriously attacked, deranged, or impaired." Accidents certainly attack, impair, and derange bodily health as effectively as bacteria.

We are justified, then, in considering accidents and the resulting injury

from the epidemiological approach, and must find how we can apply the basic epidemiological principles to this problem.

Fundamentally the four chief principles are:

1. Determination of existing status and behavior of the disorder in the community or universe being investigated.
2. Determination of causes related to the status of the specific disorder.
3. Development of specific measures for adequate control.
4. Evaluation of results that come from recommended procedures.

An example of one facet of numbers 1 and 2 already related to this problem and explored to some extent, is the concept of "accident proneness." It has been conclusively shown that certain persons are far more likely to have accidents than others, and industry has for several years used this knowledge to its productive advantage. Physical as well as psychologic and/or psychiatric factors are frequently found of etiologic significance in the presence and persistence of this trait.

It is much more difficult to identify accident-prone persons, however, than accident-prone circumstances, yet the latter, particularly in the home, has been neglected almost entirely.

The need is apparent for intensive research activity to study and clarify the home-accident pattern and for forceful educational activities along the lines indicated by these studies. It is also obvious that additional research to determine mental and emotional causes of home accidents is essential as a basis for a more complete and constructive home safety program.

A program of study along these lines has been initiated through the Out-Patient Department of the Children's Hospital of the District of Columbia.

In the meantime, before we have more specific directional information, is there any prophylactic measure that we as pediatricians can use to reduce morbidity and mortality from accidents among our patients? Edward Press of Chicago, an enthusiastic and capable student of accidents in children, points out that there is—in the form of home safety check lists in connection with home accidents and immunizations. He points out that the normal distress of mothers at the physical pain and struggling of infants during inoculation against the various communicable diseases can be capitalized on. Whenever these services are rendered, there is often the stated or implied wish of the mother to avoid the infant's suffering at the expense of her own. "If I could only take the injection for you," is not an uncommon remark. This would be an excellent opportunity to suggest that here is one form of immunization that she can take for the baby. That is, she can prevent injury or death from accidents which by this time, 4 to 8 months, constitute the chief cause of death. A thorough check

list on home safety (obtained free from the Health Department or the Safety Council) could be given to the mother at this time to be taken home and filled out carefully. Then on her subsequent visit the following month, the completed list would be reviewed by the office nurse or physician. This procedure could also be easily adapted for well-baby clinics.

Additional attempts must be made to stimulate, at community and national levels, an active appreciation of the problem of accidental death and crippling in children and an awareness that the problem is not insoluble.

SHIGELLOSIS WITH CENTRAL NERVOUS SYSTEM INVOLVEMENT SIMULATING POLIOMYELITIS

Case Report No. 219

David L. Simon, M.D.
Joseph M. LoPresti, M.D.

This four-year old white male entered the Children's Hospital on September 3, 1951 with the chief complaints of listlessness, fever, and diarrhea of one day's duration. On the day prior to admission he refused to eat lunch and slept most of the afternoon. That evening a fever of 104.0 F. rectally developed. The patient was seen in the Out-Patient Department and was found to have bilateral otitis media and pharyngitis. Therapy consisted of procaine penicillin 300,000 units daily, aspirin, and phenobarbital. On the day of admission a mild diarrhea developed and the child became extremely lethargic. Twitchings of the hands and stiffness of the neck and back became manifest and he was admitted to the hospital.

The patient's paternal and maternal grandfathers were diabetics. There was no known contact with any contagious disease.

Physical examination on admission revealed a well developed, fairly well nourished, semicomatose, four-year old white male with pallor and dry skin who appeared to be acutely ill. Temperature was 104.4 F. rectally. There was a nystagmus present but the fundoscopic examination was normal. Both external auditory canals were injected and the pharynx was markedly inflamed. The lungs and heart were normal except for a soft, high-pitched, apical, systolic murmur. The liver edge was just palpable below the right costal margin. Neurological examination revealed a 2-plus nuchal rigidity and back rigidity. The cremasteric reflexes were absent and the right patellar reflex was absent.

Laboratory work-up on the day following admission included a hemo-

gram which disclosed 13.2 grams of hemoglobin; 4,200,000 red blood cells; 3,800 white blood cells with 61 neutrophiles (49 bands, 1 segmented, and 11 young forms) and 39 lymphocytes (19 atypical and 20 mature forms) per 100 cells. Two days later, the total white count revealed 4,300 cells with 43 segmented cells, 18 band forms, 28 lymphocytes, 2 eosinophiles and 1 basophile per 100 cells. On September 11, the hemoglobin count revealed 11.2 grams; the red blood cells numbered 3,900,000; the white blood cells, 11,500 with 47 segmented forms, 6 band forms, and 47 lymphocytes per 100 cells. Admission spinal fluid was found to be clear and to contain 15 milligrams per 100 ml. protein, 45 milligrams per 100 ml. sugar, and 25 white blood cells per cubic millimeter of which 66 per cent were polymorphonuclears and 34 per cent lymphocytes. Re-examination two days later showed the fluid still to be clear; protein was 13 milligrams per 100 ml.; sugar, 45 milligrams per 100 ml.; and white blood cells 13 per cubic millimeter of which 80 per cent were polymorphonuclears and 20 per cent, lymphocytes. The non-protein nitrogen on admission was 29.6 milligrams per 100 ml.; sugar, 100 milligrams per 100 ml.; and the carbon dioxide combining power was 37 volumes per cent. Admission stool culture was positive for *Shigella flexneri*.

After the admission spinal puncture was performed, the patient was started on chloromycetin palmitate and sulfadiazine. Intravenous fluids were initiated, and within eight hours, the patient's temperature dropped from 104.4 F. rectally to 99.0 F. rectally. He had four, semi-solid stools on the day of admission and was placed on a diarrhea diet and boiled skimmed milk. Stool cultures taken on September 2, 3, 4, and 6 grew out Shigella organisms of the flexner type.

On September 7, 1951 a three-year old sister of the patient entered the hospital with irritability and a history of vomiting three times and of having four semi-liquid stools. Spinal fluid was essentially normal, but stool cultures similarly grew out *Shigella flexneri*.

On September 9, 1951, the patient's father was hospitalized elsewhere as a possible meningitis. Spinal puncture was negative. All cultures were negative and a diagnosis of grippe was made. The patient's mother and another sibling were placed on chloromycetin prophylactically.

Stool cultures on both children admitted to Children's Hospital became negative on the fourth day and remained free of pathogens for three consecutive days. Both patients were discharged on September 13, 1951 as completely recovered. Neurological examination on discharge was reported as normal.

DISCUSSION

The differential diagnosis of poliomyelitis often poses a problem. During the summer months, many patients, admitted to the hospital as suspected

cases, subsequently are proven to have a condition which simulated poliomyelitis. Among the diseases which mimic poliomyelitis, bacillary dysentery is singularly neglected as a diagnostic possibility. The problem is further complicated because diarrhea is a frequent concomitant of poliomyelitis, and meningismus may accompany bacillary dysentery.

The specific enteric infections are world-wide in their distribution. Shiga first described the causative agent in Japan in 1898; and in 1899 in the Philippines, Flexner isolated the etiologic agent which now bears his name. The term "shigellosis" is a generic one and includes bacillary enteric infections, with or without dysentery, caused by three main groups of Shigella organisms. These groups may be differentiated by their ability to ferment sugars.

I. *Shiga group:* does not ferment mannite and possesses a high specific antigenicity. A neurotropic exotoxin has been demonstrated for this group. Most of the Shigella strains cause a severe illness. In the United States, this group is responsible for less than 5 per cent of the dysenteries.

II. *Flexner group:* ferments mannite but not lactose. This large group of organisms varies somewhat in its serological reactions and ability to ferment sugars other than mannite. Flexner infections may be mild or malignant and cause at least 80 per cent of the dysenteries in America.

III. *Sonne-dispar group:* ferments both mannite and lactose. Infestation is usually productive of mild illness, and it is probable that from 10 to 20 per cent of Shigella infections are due to this group.

The usual patient with Shigellosis (bacillary dysentery) presents a rather typical clinical picture. The disease may be mild or severe depending on the height of the fever, the amount of diarrhea, the degree of acidosis and dehydration, and the duration of illness. Unusual types have been described and, in the descriptions of various outbreaks, neurological manifestations have occurred in a significant percentage of the patients. Dodd, Buddingh, and Rapaport[1] studied the etiology of Ekiri, a highly fatal form of enteritis peculiar to Japan. They proved the Ekiri and Shigellosis were, for the most part, synonymous. Convulsions occur in most of the patients with Ekiri. However, a low serum calcium was indited as the cause of the neurologic symptoms in the described outbreak. Hardy and Watt[2, 3] reported convulsions in less than 3 per cent of the patients studied and mention meningismus as an accompanying symptom in an occasional patient with bacillary dysentery. Yet, they do not include meningitis in the differential diagnosis. Felsen and Osofsky[4] reported 8 cases of Sonne dysentery and central nervous system symptoms were not noted in any instance. In 1937, an outbreak occurred in Jersey City[5]. In 100 patients studied, 13 per cent showed a stiff neck and positive Kernig's and Brudzinski's signs. In all instances, the spinal fluid was normal. Tandeta and Twible[6] reported 2 patients admitted during the

height of the poliomyelitis season as suspicious because of the presence of neurological manifestations. In both children, the spinal fluid was normal and the true nature of the illness was manifested within twenty-four hours. In discussing bacillary dysentery, Lyon[7] states that in some cases, the diarrhea does not appear at once, and the disease may have its onset with stupor, delirium, stiffness of the neck, and even convulsions. The clinical picture may resemble that of meningitis or encephalitis. As the case progresses, however, the intestinal symptoms come more and more into prominence, and the cerebral symptoms usually subside. Shaeffer[8] notes that dysentery may at times be confused with meningitis and in some cases a differential diagnosis is impossible except by lumbar puncture. Bradford[9] affirms that in the prediarrheal stage of dysentery, meningeal symptoms are frequently present. Meningitis or the preparalytic stage of poliomyelitis may be suspected, and a lumbar puncture may be necessary to eliminate them as diagnostic possibilities.

Most of the authorities agree, therefore, that the diagnosis of dysentery may be masked by neurological symptoms and at times a lumbar puncture may be necessary to exclude central nervous system disease. It is assumed that the spinal fluid in patients with Shigellosis will be normal. No case report could be located in the medical literature in which alterations of the spinal fluid are described. Our patient was exceptional in this respect. It is unlikely that the cerebrospinal fluid pleocytosis represented the presence of another disease process, e.g., poliomyelitis or encephalitis. The course of the illness, the absence of muscular spasm or weakness, and the rapidity with which the abnormal spinal fluid findings returned to normal would seem to exclude these pathological entities. It is more likely that all of the neurological manifestations were an expression of the same disease process, i.e., infestation with *Shigella flexneri*. If this be true, and cerebrospinal fluid pleocytosis, on occasion, may be part of the clinical picture of Shigellosis, then lumbar puncture, in some patients, would be insufficient to differentiate bacillary dysentery from central nervous system disease, particularly encephalitis or poliomyelitis. It will be necessary to perform further laboratory examinations on such patients to ascertain the true nature of the illness.

SUMMARY

1. An unusual case report is presented of a four-year old white male with bacillary dysentery caused by *Shigella flexneri* in which neurological symptoms and a cerebrospinal fluid pleocytosis were present. This is thought to be the first case of Shigellosis in which spinal fluid alterations are recorded.

2. Bacillary dysentery (Shigellosis) should be included in the differential

diagnosis of poliomyelitis. If changes in the spinal fluid can occur during the course of Shigella infestations, then lumbar puncture is insufficient to exclude central nervous system disease as a diagnostic possibility. In such cases further clinical evaluation will be necessary.

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CONGENITAL URETHRAL VALVE WITH APPARENT ASCITES

Case Report No. 220

Robert H. Parrott, M.D.

INTRODUCTION

The following case of congenital urethral valve obstruction is presented for two reasons:

1. The interesting initial clinical picture of apparent ascites and pitting edema of the lower extremities in a newborn infant is rare and

2. The case serves to emphasize again the relative frequency of urinary tract abnormalities in pediatric practice.

Case Report B. P. 50-10837

B. P., an eight-day old colored male was admitted to the hospital with the chief complaints of loss of appetite and a distended abdomen. His gestation and birth had been normal. The birth weight was six pounds, twelve ounces. He was taken home from the maternity hospital at three days of age, and his parents began to note that he ate very little and that his abdomen was becoming progressively dis-

tended. The child was said to have had normal urination and defecation. There was no jaundice, vomiting or diarrhea. The abdominal swelling increased to such an extent that the patient's father had noted a visible venous pattern on the anterior abdominal wall two days prior to admission.

Family history was non-contributory.

On physical examination (c.f. Figure 1), it was noted that a marked abdominal swelling and pitting of the lower extremities were present. The infant cried lustily and had an intact Moro response. The head, eyes, ears, nose and throat were normal.

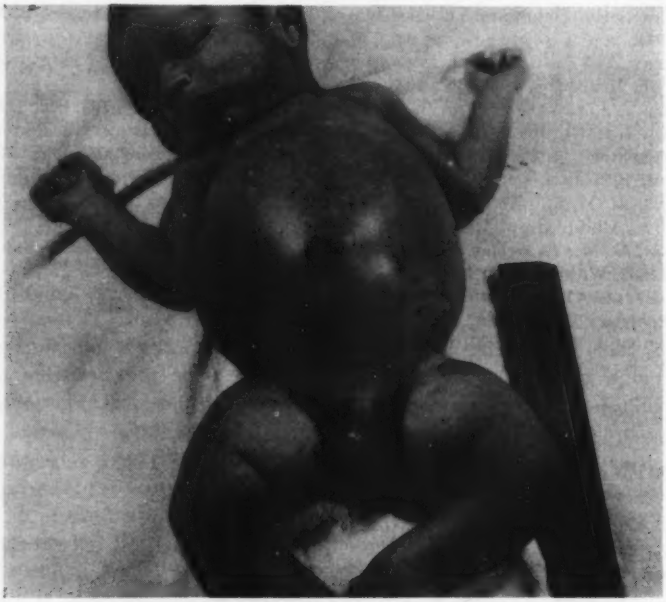

FIG. 1

The lung fields, although compressed by the abdominal swelling, were normal. The heart sounded normal. Adequate blood pressure determinations could not be made at that time.

The abdomen was 52 centimeters in girth and was markedly distended with some degree of pitting edema of the skin which did not extend above the costal margins; the umbilicus was slightly reddened. Due to the distension, there was a visible venous pattern and a suggestive caput medusae on the anterior abdominal wall. A fluid wave was elicited. Light could be transmitted through most of the abdomen except the postero-lateral aspect of each upper quadrant. There was a 2 plus pitting edema of both lower extremities. A tentative diagnosis of inferior vena-caval obstruction was made and the child was admitted for study.

On the day after admission, the infant developed dyspnea and signs of diminished circulation in the lower extremities. An attempt to catheterize the bladder was made

without success. Abdominal paracentesis yielded 350 milliliters of slightly turbid, yellow serous fluid with a specific gravity of 1.002. It was found to be sterile but to contain acute and chronic inflammatory cells and a large number of squamous-appearing cells. A moderate degree of distension persisted. The possibility arose that the fluid withdrawn was urine rather than ascitic fluid.

Liver function tests, total proteins, and peripheral blood counts were normal, but the non-protein nitrogen was 59 milligrams per 100 milliliters. When a barium enema showed a circumscribed mass in the bladder area, further attempts at cathe-

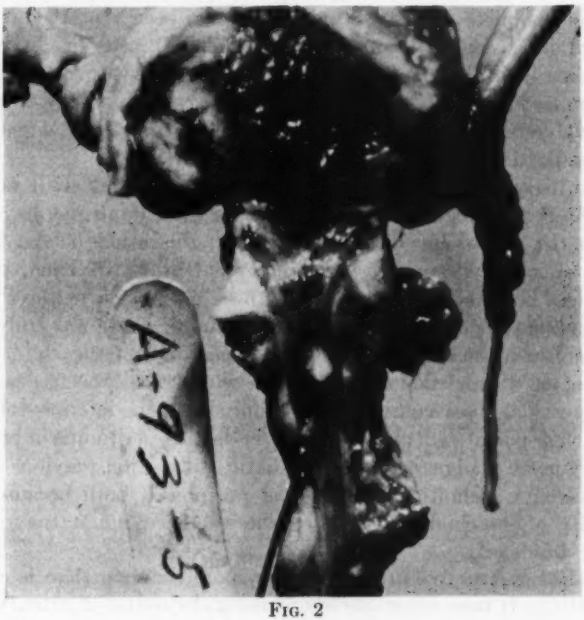

FIG. 2

terization, bypassing an apparent posterior urethral obstruction, yielded 375 milliliters of clear yellow urine. This had a specific gravity of 1.010 and showed only rare white blood cells per high power field. It was sterile on culture. The abdomen was now 46 centimeters in girth. An indwelling catheter was inserted and the child was placed on aureomycin and penicillin. Intravenous pyelograms showed only minimal filling of the left kidney in three hours. After consultation with the urological service, it was decided to manage the baby on catheter drainage as necessary, following the non-protein nitrogen determination, to ascertain whether there would be any recovery of renal function. Subsequent cystoscopy or suprapubic drainage was contemplated.

The child's course, however, was gradually downhill; bilateral flank masses became palpable. Despite drainage and antibiotics, there was continued elevation of the non-protein nitrogen to 61 milligrams per 100 milliliters; a pyuria developed.

and the abdominal distension persisted. Edema of the legs intermittently improved and regressed. The child expired twenty days after admission.

Post mortem examination confirmed the presence of a congenital urethral valve (c.f. Figure 2). There was bilateral megalo-ureter, chronic pyelonephritis, and pyonephrosis and hypertrophy of the bladder wall. An incidental finding was fibrosis of the pancreas with duct dilatation. Pseudomonas aeruginosa was cultured from the blood, cisterna magna fluid, and bladder urine.

DISCUSSION

Cases of complete urethral obstruction have been reported with such abdominal distension as to interfere with delivery[1]. Stevens[2] records an instance in which bladder distension was associated with marked ascites and generalized edema. Usually, however, the symptomatology of posterior urethral-valve obstruction, while present from birth, does not reach such morbid proportions as in this child until later infancy.

Since urinary secretion begins between the fifth and sixth month of fetal life[3], the pathogenesis of the disease in this infant may have begun some months before his birth. Presumably, dilatation of the bladder, hydroureter and hydronephrosis, were present at birth. Then, with the added stress of independent existence, these pathologic features became more prominent, as noted by the parents. Whether there was truly ascites shall remain a moot question—but the appearance of the child and the presence of leg edema led observers to believe that there was free peritoneal fluid. Other diagnoses considered at admission were inferior vena-caval obstruction or portal-vein obstruction, liver disease, peritonitis or peritoneal irritation due to malignancy and lymphatic obstruction (chylous ascites). As noted above, definitive surgery was postponed, both because of the immaturity of the child and to determine whether renal status would improve on drainage.

Congenital obstructive uropathy is far more common than is clinically suspected[4, 5]. It may occur anywhere along the course of urinary flow in the form of stenosis, valves, tumor, redundancy, or external pressure, e.g., from an anomalous vessel. Among the most common of such lesions are posterior urethral valves which occur almost exclusively in males. These are mucosal redundancies, usually multiple, which extend from the verumontanum to the bladder outlet, to the lateral wall or to the membranous urethra[3]. They may be cusps or may assume a diaphragm shape. They float into an obstructing position when voided urine meets them and thus initiate the entire chain of events leading to the picture of obstructive uropathy and chronic pyuria.

Etiologically, they may be embryological remnants of the urogenital membrane[6]. The symptomatology of such lesions are frequency, dysuria, hesitancy, recurrent pyuria, and hematuria. Signs of uremia may eventually supervene.

The definitive diagnosis may be made by endoscopy[3], visualizing the cusp-like action of the vales in an irrigating stream. The diagnosis may be suspected from a history of recurrent pyuria and obstructive symptoms, from radiographic signs of hydronephrosis, and enlarged bladder and by occasional difficulty in catheterization.

According to Campbell[3], the only satisfactory treatment is transurethral resection of the valves. Others advocate disruption of the valves with sounds or suprapubic and perineal excision of valves.

SUMMARY

1. A case is presented of urethral-valve obstruction in a newborn with massive abdominal swelling and leg edema.
2. The nature of this illness is discussed.

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SUBJECT-AUTHOR INDEX FROM DECEMBER 1950 THROUGH DECEMBER 1951

Subject Index

Author Index

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